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Effect of NHIP on reactive oxygen species levels in MeCP2^{+/-} neural progenitor cells

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Abstract:

Oxygen is critical for proper brain function. During hypoxia, insufficient oxygen levels can increase the level of reactive oxygen species (ROS), damaging neurons and their DNA. Our lab discovered *NHIP* (neuronal hypoxia inducible, placenta), a gene that encodes a short peptide that may protect neurons from hypoxia-induced stress. When exogenous NHIP peptide was added to a neuronal cell line 24 hours prior to 1% hypoxia exposure, ROS levels decreased. In a screen for proteins that interact with NHIP, we identified methyl-CpG-binding protein 2 (MeCP2) as a potential binding partner. Loss of MeCP2 leads to Rett Syndrome (RTT), and many RTT patients display breathing abnormalities that lead to intermittent hypoxia. We hypothesize that the interaction between NHIP and MeCP2 promotes proper hypoxia response. Preliminary experiments show that NHIP decreases ROS levels in neural progenitor cells isolated from wild-type mice, but not in neural progenitor cells isolated from MeCP2^{+/-} mice, which have dysfunctional MeCP2 protein. This suggests that proper MeCP2 function is necessary for the reduction of ROS levels by NHIP. Characterization of NHIP and the mechanism by which it reduces ROS levels will allow for evaluation of its potential use in protecting neurons from injury under hypoxic conditions.

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